

PAROTID TUMOURS

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TUMOURS of the salivary glands are relatively uncommon. From 1958 through 1968 a total of 77 lesions of all the salivary glands were seen by one of us (M.B.) and 67 involved the parotid, 8 the submaxillary, one the sublingual and one an ectopic gland. In this paper we present our experience with the 'partoid' lesions, which comprise the largest group in the series, 67 out of a total of 77, as enumerated in Table I.

CLINICAL FEATURES

The classical and frequently the only symptom and sign of a parotid tumour is a swelling. Only occasionally is slight pain or discomfort noted. Very often there is a long delay from the time the swelling is first noticed until the patient is referred to hospital, and in this series it varied from six weeks to 38 years.

Of the 7 patients with carcinoma of the parotid, 5 had local pain. Some degree of facial paresis is often mentioned as an early sign, but in this series 5 out of 7 cases had no facial paresis when first seen. So far as could be ascertained, 4 patients had no pre-existing tumour, while in the remaining three there had been a pre-existing tumour, presumably benign, for several years. In one instance a tumour had been present for about 30 years, but two months before referral to hospital it began to increase in size, the patient developed pain and there was weakness of the facial nerve.

DIFFICULTY IN DIAGNOSIS

Accurate pre-operative diagnosis of a swelling in the parotid gland is difficult. Patey (1968) quoted Bland Sutton, who said that swellings in the parotid were "interesting lumps that required removal." In some cases the history is helpful, and in a few the diagnosis is comparatively easy. Of the lesions listed in Table I, acute non-purulent parotitis, chronic parotitis and calculus are fairly readily diagnosed. Also cystic hygroma and branchial cyst should be suspected from the clinical features. In all the other lesions seen diagnosis was not possible from the

TABLE I — *Parotid Tumours* — 67

Cystic Hygroma	1	Acute (non-purulent parotitis	2
Bronchial Cyst	2	Chronic Parotitis	16
Lipoma	4	Calculus	2
Boeck's Sarcoidosis	1	Mixed Salivary Tumours	24
Hodgkin's	1	Adenoma	2
Sec. Carcinoma	1	Adeno-Lymphoma	2
		Carcinoma	7
		Carcoma	2

history and clinical examination, with the possible exception of the one case of secondary carcinoma. The frequency of wrong clinical diagnosis is high and in a study carried out some years ago it amounted to 40 per cent (Patey and Hand, 1952).

Sialography has not been found of any help in diagnosis. The question therefore of biopsy has to be considered. It is well known that mixed salivary tumours (pleomorphic salivary adenoma) have a very thin capsule which is easily ruptured with resultant spillage of tumour cells into adjacent normal tissue. This can result in the production of multiple tumours in the parotid, or indeed, in the overlying skin. The danger of implantation is less with needle biopsy but has occurred (Eneroth, 1964). Interpretation of such limited material is difficult on histological examination.

Wide excisional biopsy is therefore necessary. That is, removing a wide margin of normal tissue with the tumour. This raises special problems in tumours of the parotid because of the intimate relationship of the trunk and branches of the facial nerve to the parotid. Excisional biopsy therefore involves exposure of the trunk of the facial nerve and subsequent identification of its main and subsidiary branches. At least 90 per cent of tumours of the parotid gland arise in the superficial lobe and therefore excisional biopsy essentially means superficial parotidectomy. There should be no resultant paresis of the facial nerve.

TREATMENT

The main principles of treatment of tumours of the parotid gland can be summed up as – wide surgical exposure by a cervico-facial (Y-shaped incision), early identification of the trunk of the facial nerve and removal of the tumour together with such surrounding tissues as the pathology demands (Patey, 1966).

It is generally agreed that it is better in the majority to expose the facial nerve as it emerges from the stylomastoid foramen than to trace back one of its branches from the anterior border of the parotid. This latter method results in a high incidence of residual permanent paralysis of the facial nerve. The nerve runs outwards and forwards from the stylomastoid foramen and at the isthmus of the parotid gland divides into two main branches which subsequently divide into the terminal branches of the nerve (*pes anserinus*). Removal of the gland superficial to the facial nerve is known as superficial parotidectomy. Removal of the whole of the parotid, sparing the facial nerve is termed total parotidectomy. Radical parotidectomy means total parotidectomy with sacrifice of the facial nerve.

The importance of the venous plexus lying immediately deep to the facial nerve has been emphasised (Patey and Ranger, 1957). Troublesome venous bleeding can occur from one of these small veins and anaesthesia with induced hypotension has been found to be of help in what can sometimes be a difficult dissection, particularly in cases of chronic parotitis.

Local excision of a small peripheral tumour is possible because, in such a situation the tumour can be excised with a margin of normal parotid tissue without the risk of significant damage to the facial nerve. In treating malignant tumours, radical parotidectomy is necessary in the majority of cases. In a very small lesion it may be possible to preserve one of the main divisions of the facial nerve. Where there is involvement of lymph glands in the neck block dissection is indicated with

radical parotidectomy. Most malignant tumours are not sensitive to radiation but it has been employed following one or two difficult dissections where it was considered that there was spillage of the tumour during operation. The various methods of treatment are indicated in Table II, and more specific reference will be made to pre-operative regional intra-arterial infusion.

TABLE II — *Method of Treatment*

Benign Tumours	(a) Superficial Parotidectomy ? ? Local Excision
	(b) Total Parotidectomy
Malignant Tumours	(a) Radical Parotidectomy
	(b) Parotidectomy with partial preservation of facial nerve
	(c) Radical Parotidectomy and Block Dissection
	(d) Pre-operative regional intra-arterial infusion and radical parotidectomy
	(e) Radical Parotidectomy and Deep X-ray Therapy

Mixed Parotid Tumours (Pleomorphic Salivary Adenomas)

In the series superficial parotidectomy was performed in 16 cases. Total parotidectomy with conservation of the facial nerve was carried out in one case. This was necessary because the tumour involved the deep part of the gland. Local excision was performed in 3 cases. In 4 cases the tumour was recurrent following operation elsewhere. The recurrences were 1 year, 4 years, 16 years and 33 years respectively after the primary operation. Three of these were treated by superficial parotidectomy and one by local incision.

In the series radiotherapy was given post-operatively in two cases where spillage occurred during the operation.

Malignant lesions of the Parotid

There were 7 cases of carcinoma and two cases of sarcoma, as detailed in Table III. In the carcinoma group, two cases with mucoepidermoid tumours were seen at a comparatively late stage when they had involvement of glands in the neck, and were treated by radical parotidectomy with ipsilateral block dissection of the cervical glands. Likewise, one patient with an acinic cell tumour required radical

TABLE III — *Malignant Group*

<i>Carcinoma</i>		<i>Sarcoma</i>	
Mucoepidermoid	2	Reticulum Cell	1
Acini Cell	1	Myosarcoma	1
Squamous	2		
Adenocarcinoma	1		
Undifferentiated	1		

parotidectomy with ipsilateral block dissection of the cervical glands and one of the cases had post-operative radiotherapy. Of the two cases of squamous carcinoma, one had a very small tumour and was treated by superficial parotidectomy, the other case had had a biopsy done elsewhere and the tumour seemed to be growing very rapidly, with a very marked inflammatory reaction, so that surgical treatment was not considered possible. We had recently treated an elderly patient with an inoperable recurrent squamous carcinoma of cheek by regional intra-arterial retro-grade infusion of methotrexate via the superficial temporal artery, and were greatly encouraged by an initial dramatic response. Although this was not maintained it did encourage us to try the same treatment, in this case of carcinoma of the parotid. There was a most encouraging response as evidenced by regression in size of the tumour and disappearance of all the inflammatory reaction, at which stage we proceeded to do a parotidectomy. This patient did not have facial paresis pre-operatively but at operation it was found impossible to preserve the trunk and upper branches of the facial nerve. The case of undifferentiated carcinoma was treated by superficial parotidectomy in the first instance, because the tumour was small and there was no involvement of the facial nerve. Subsequently, when we received the histological report this patient was referred for post-operative radiotherapy.

There were two cases of sarcoma of the parotid, the case of reticulum cell sarcoma had a small tumour and was treated as though he had a pleomorphic salivary adenoma. When the histo-pathology was subsequently revealed he was referred to for post-operative x-ray therapy. The case of myosarcoma had had one or two biopsies done elsewhere and had a most extensive tumour with involvement of the cervical glands on the affected side. He was treated by radical parotidectomy and ipsilateral block dissection of the cervical glands.

COMPLICATIONS

Complications are not frequently encountered and are as detailed in Table IV. Infection has been noted in several cases but has never been severe or persistent, with one exception, a case of intractable chronic parotitis. The patient inexplicably developed thrombosis in the vessels in the lobe of his ear which subsequently proceeded to a state of dry gangrene and separated.

TABLE IV — *Complications of Operation*

Infection	Mild in four cases
Haematoma	Two cases
Sensory loss	In lobe of ear in all cases
Facial paresis	Partial and temporary in 8 cases Total and permanent in 3 cases. Total and permanent in 3 cases of carcinoma and one case of sarcoma Residual weakness in 2 cases of recurrent mixed salivary tumour
Auriculo-temporal syndrome	2 cases
Parotid Fistula	None

In this series it was not found possible to preserve those branches of the great auricular nerve going to the lobe of the ear, and thus there is a loss of sensation in the lobe of the ear in all cases. Patey states that it is possible to preserve these branches in some cases but to date it has not been found possible by us. The loss of sensation in the lobe of the ear has been commented upon by many of the patients, but has not otherwise been a problem.

Regarding facial paresis, the importance of identification of the trunk and branches of the facial nerve must be stressed. It will be noted that in three cases there was temporary and total facial paralysis, but when the operator has seen the facial nerve, he can with confidence reassure the patient and, in our three cases, there was subsequent complete restoration of function. The interval was 1 month, 2 months and 3 months respectively. The branch of the facial nerve which is most often involved is perhaps the longest branch, that is, the one supplying the angle of the mouth and the lower lip. Paresis involving this branch of the nerve has only persisted in two cases who had recurrent mixed salivary tumours.

The auriculo-temporal syndrome did appear in two cases, and it is interesting to note that in one case it did not appear for approximately one year following superficial parotidectomy, and in the other, several months after operation. It has not been severe in either case and in the first case it has clear up almost completely.

RESULTS

All cases have had complete follow-up to date. Table V summarises the results in the mixed parotid and recurrent mixed parotid tumours. Table VI shows the

TABLE V — *Mixed Tumours*

<i>Type</i>	<i>No. of cases</i>	<i>Follow-up in years</i>			
		1	4	8	
Primary	20	10	4	1	
Recurrent	4	3	1	—	
Total	24	13	5	1	

There have been no recurrences in the follow-up periods.

TABLE VI — *Carcinoma*

<i>Type</i>	<i>No. of cases</i>	<i>Follow-up in years</i>		
		6/12	1 year	2 years
Squamous	2	(1)	—	(1)*
Adenocarcinoma	1	—	(1)	—
Undifferentiated	1	—	1	—
Mucoepidermoid	2	(2)	—	—
Acinic Cell	1	—	—	1

Parenthesis show recurrences and death from metastasis.

*Died at 2 years from heart attack — no evidence of disease.

results in seven cases of carcinoma broken down into histological types. The two cases with adenolymphoma show no evidence of recurrence 7½ years and nine months post-operatively.

DISCUSSION

Tumours of the parotid are uncommon and as has been pointed out accurate pre-operative diagnosis as to the nature of the pathology is difficult. With the wider acceptance of the principles of parotid surgery treatment is becoming more standardised. For mixed tumours superficial parotidectomy is the treatment of choice. Radiotherapy has little or no effect on the primary tumour but can be used post-operatively where spillage has occurred in an attempt to destroy microscopic tumour deposits. Evidence for this is that in the past, when surgical treatment was more conservative and tumours were simply shelled out, the implantation of radium was found to reduce the incidence of recurrence (Ahlbom, 1935).

Van Meirt, Dawes and Harkness (1968) recommended simple enucleation followed by radiotherapy in the treatment for mixed parotid tumours. Over a period of twenty-six years a total of 167 patients were treated by enucleation and radiotherapy and recurrence rate was stated to be 2.74 per cent, but this method of treatment is not generally accepted.

Follow up of these cases has to be long as late recurrence is a characteristic (Patey, 1967). Recurrent mixed tumours are more difficult to treat because although clinically they may appear to be a single nodule, there is often widespread diffusion of tumour in the operation area. The risk of damage to the facial nerve is greater and in the present series two cases have residual weakness of the lower part of the facial nerve.

In most series the percentage of tumours classified as malignant is in the order of 15 to 25 per cent (Patey et al, 1965; Eneroth, 1964; Sharp and Helsper, 1960), but an incidence of 34 per cent was reported by Foote and Frazell (1954). In the present series it was 16.3 per cent. The results of treatment of carcinoma are bad, most patients dying of the disease irrespective of the treatment given. One case in this series who had an inoperable squamous carcinoma when first seen and to which reference has already been made (*vide supra*) had a dramatic response to regional intra-arterial retrograde infusion of methotrexate. This produced a situation in which surgery was possible, and the patient lived for two years and died from a heart attack.

Chemotherapy in the treatment of malignant disease has most often been used for late cases when other methods of treatment have failed. The head and neck is one area where regional infusion is anatomically feasible, and while one cannot draw conclusions from encouragement in one case it does, nevertheless, suggest that pre-operative infusion of a chemotherapeutic agent should be tried in an area where our present methods of treatment give unsatisfactory results.

SUMMARY

All cases of parotid tumours occurring over a ten year period are reviewed with particular reference to clinical features, difficulty in diagnoses, treatment, complications and results.

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BOOK REVIEW

SOME INHERITED DISORDERS OF BRAIN AND MUSCLE. Edited by J. D. Allan and D. N. Raine. (Pp. viii+154; figs. 53. 40s). Edinburgh and London: E. & S. Livingstone, 1969.

THIS volume is the published proceedings of the fifth symposium of the Society for the Study of Inborn Errors of Metabolism held in Newcastle in 1968. Previous symposia in this series have been valuable collections of papers in a rapidly expanding, multi-disciplinary field and this volume is no exception. Walton's discussion of the classification of muscular dystrophy is a fair summary of a difficult subject. Some of the papers are of specialist interest e.g. McArdle's on skeletal muscle glycogenosis, but because of the combined clinical and biochemical discussion even these are of general importance. The cerebral lipidoses feature prominently in the papers on disorders of brain metabolism, presumably because other abnormalities have been discussed in previous symposia, but Walshe's combined genetic and biochemical study in Wilson's disease helps to balance this part of the book.

Obviously this volume has a restricted appeal but it contains information of great interest to many medical specialists and can be recommended to the biochemist, the morbid anatomist and the paediatric neurologist. To those contemplating postgraduate examinations it provides valuable information on some of the rare disorders which seem to interest some examiners.

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